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ANSWER 1 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER:

2001:759961 CAPLUS 136:161034

DOCUMENT NUMBER: TITLE:

Anti-TNF- α Properties of New 9-Benzyladenine Derivatives with Selective Phosphodiesterase-4-

Inhibiting Properties

AUTHOR(S):

Reimund, Jean-Marie; Raboisson, Pierre; Pinna,

Guillaume; Lugnier, Claire; Bourguignon, Jean-Jacques;

Muller, Christian D.

CORPORATE SOURCE:

Laboratoire de Pharmacologie et Physico-Chimie des Interactions Cellulaires et Moleculaires, UMR 7034 du CNRS, Universite Louis Pasteur de Strasbourg, UFR de Sciences Pharmaceutiques, Illkirch, 67401, Fr.

SOURCE:

Biochemical and Biophysical Research Communications (

2001), 288(2), 427-434 CODEN: BBRCA9; ISSN: 0006-291X

PUBLISHER: Academic Press

DOCUMENT TYPE: Journal LANGUAGE: English

In inflammatory cells, intracellular cAMP concentration is regulated by cyclic nucleotide phosphodiesterases 4. Therefore, PDE4 inhibition appears as a rational goal for treating acute or chronic inflammatory diseases. Selective PDE4 inhibitors have been developed, but due to unwanted side effects, search for new selective PDE4-inhibitors had to be pursued. Recently, Boichot et al. (J. Pharmacol. Exp. Ther. (2000) 292, 647-653) showed that 9-benzyladenine derivs. are selective PDE4 inhibitors. In vivo data in animals suggested that they may induce fewer side effects (emesis). We examined the effects of new 9-benzyladenines on TNF- α , interleukin (IL)-1 β , IL-6 and IL-8 production by lipopolysaccharideactivated peripheral blood mononuclear cells, and compared them to other PDEs inhibitors. Selected potent 9-benzyladenines, strongly inhibited TNF- α production Interleukin-1 β , IL-6, and IL-8 production was not significantly affected. Our results suggest that some of these new adenines (i.e., NCS 675 and NCS 700), may be potential therapeutic candidates for the treatment of inflammatory diseases. 2001 Academic Press.

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ANSWER 2 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN.

2001:670031 CAPLUS ACCESSION NUMBER:

DOCUMENT NUMBER: 136:31471

Urodilatin, a natriuretic peptide stimulating TITLE:

particulate guanylate cyclase, and the

phosphodiesterase 5 inhibitor dipyridamole attenuate

experimental pulmonary hypertension. Synergism upon

coapplication

Schermuly, Ralph Theo; Weissmann, Norbert; Enke, AUTHOR(S):

Beate; Ghofrani, Hossein Ardeschir; Forssmann, Wolf

Georg; Grimminger, Friedrich; Seeger, Werner;

Walmrath, Dieter

CORPORATE SOURCE: Department of Internal Medicine, Justus-Liebig-

University Giessen, Giessen, D-35392, Germany American Journal of Respiratory Cell and Molecular SOURCE:

Biology (2001), 25(2), 219-225 CODEN: AJRBEL; ISSN: 1044-1549

American Thoracic Society PUBLISHER:

Journal DOCUMENT TYPE: English LANGUAGE:

In a model of acute pulmonary hypertension in intact rabbits, the authors investigated the vasodilatory potency of intravascularly administered urodilatin, a renal natriuretic peptide type A known to stimulate particulate quanylate cyclase. Urodilatin infusion was performed in the absence and presence of the phosphodiesterase (PDE) type 5 inhibitor dipyridamole. Stable pulmonary hypertension was evoked by continuous infusion of the thromboxane mimetic U46619, resulting in approx. doubling of the pulmonary artery pressure (PAP). When infused as sole agents, both urodilatin and dipyridamole dose-dependently attenuated the pulmonary hypertension, with doses for a 20% decrease in PAP being 30 ng/kg min for urodilatin and 10 µg/kg min for dipyridamole. A corresponding decrease in systemic arterial pressure (SAP) was noted to occur in response to both agents. Sequential i.v. administration of a subthreshold dose of dipyridamole (1 μ g/kg min), which per se did not affect pulmonary and systemic hemodynamics, and a standard dose of urodilatin (30 ng/kg min) resulted in a significant amplification of both the PAP and the SAP decrease in response to the natriuretic peptide. At the same time, manifold enhanced plasmatic cyclic guanosine monophosphate (cGMP) levels were detected. Aerosolized dipyridamole also dose-dependently attenuated pulmonary hypertension, with only 1 $\mu g/kg$ min being sufficient for a

20% decrease in PAP, with no SAP decline. Preceding administration of subthreshold aerosolized dipyridamole (50 ng/kg min) did, however, cause only a minor amplification of the pulmonary vasodilatory response to a subsequently infused standard dose of urodilatin. In conclusion, this is the 1st study to show that urodilatin does possess vasodilatory potency in the pulmonary circulation, and enhanced blood plasma levels of cGMP and synergy with the PDES inhibitor dipyridamole both strongly suggest that this effect proceeds via guanylate cyclase activation. The effect of infused urodilatin is, however, not selective for the pulmonary vasculature, as the systemic vascular resistance declines in a corresponding fashion.

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